experienee this has heen found to be one of the most reliable methods of covering sometimes a hopeless area that would otherwise demand reseetion of the howel. If the gut is too hadly involved then indeed resection is at times necessary. Failure to take eare of these details in a painstaking way and to look out for every raw and injured area may result in a fecal fistula, an ileus, or some other trying and at times fatal complication. Nowhere in the human body is it more necessary to make every detail of the operative technique perfect than in the ahdominal cavity, for when once the work is finished and sewed up there is no way to take a daily look at what is going on, as might be done with an amputation or other external operation. The careless placing of a suture that might leave a fecal leak, or the omission to cover over a weakened place in the gut, or the slighting of what may sometimes seem a trivial detail, may destroy the work of an otherwise hrilliant operation and cost the patient her life, and this is true of all ahdominal surgery.

CEREBRAL EDEMA (WET BRAIN) IN CHRONIC ALCOHOLISM.

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Our paper concerns itself with a phase of chronic alcoholism, to whieh the pathologically prominent change has given the name cerehral edema, or wet brain.

The clinieal complex, which we purpose elaborating has been given scant place in the literature upon complieations of alcoholism; only the most cursory mention of it, in fact, is made in any publication, with exception of two American texts. Dana[1] seems to have heen the first to recognize the condition. His deseription is very complete in most details, and it is only because of our considerahle experience with wet brain at the House of Correction Hospital, Chicago, and because we presume to add to the picture hoth from the point of view of clinieal manifestations and differential diagnosis, that we reopen the subject.

Lambert[2] is the other American who deseribes cerebral edema

[1] Text-book of Nervous Diseases and Psychiatry, 7th edition, p. 444 et seq.
[2] Modern Medicine (Osler), I, p. 186, et seq.

at some length. He, too, lays stress upon the meagerness of literature relative to the subject, and credits Dana with the first recognition of this phase of alcoholism. Standard works, such as Oppenheim,[3] Lewandowsky,[4] and Curschmann,[5] speak only of a serous meningitis which may be traced, in some cases, to chronic alcoholism. This type of meningitis, however, we would emphasize at the outset, is something quite distinct from the condition which is the topic of our paper. It is true that long-continued alcoholic overindulgence may be associated with serous meningitis; the latter, however, may be secondary to a number of factors, toxic, infectious, traumatic, and mechanical, and presents clinical features indicative of gross nervous changes, such as pareses, pathological cerebrospinal fluid and the like. Alcoholic cerebral edema, on the other hand, is, to all appearances, a toxic vascular phenomenon, and its correlated clinical manifestations are not similar to those of inflammations of the brain or its coverings.

Wassermeyer,[6] in his exhaustive monograph on delirium tremens, gives no place to the train of symptoms which we look upon as characteristic of wet brain and the general absence of reference to the subject in foreign literature, brings up the question whether we may not see alcoholics under conditions differing from those generally obtainable elsewhere. About 2500 cases of alcoholism come under our observation annually. They include patients of every age, sex, and social condition; as to the last, however, the great majority are from the lowest stratum of society. Some have been subjected to trauma of one kind or another; others are suffering from an infection, acute or chronic. Most of the patients are naturally enough accustomed to the cheapest alcoholic beverages. All of these factors are present in practically equal degree in most American public hospitals, and, so far as our personal knowledge goes, in German and Austrian institutions, too, so that we feel justified in assuming that we are working under no unusual conditions.

Wet brain, dissected out from the network of alcoholic complications and sequelæ regularly associated with it, is definitely delimited in nearly every respect, particularly as to classification, symptomatology, and prognosis.

Our view as to its place in the phase of chronic alcoholism has undergone change from time to time. Certain etiological factors deserve emphasis: (1) the longer the history of alcoholism and the more frequent the attacks of delirium tremens the more likely is the patient to develop wet brain, and one attack predisposes to a second; (2) some patients regularly go over into the wet-brain state from each attack of delirium; (3) cerebral edema always

[3] Lehrbuch der Nervenkrankheiten, Berlin, 1908, II, S. 1087.
[4] Handbuch der Neurologie, Berlin, 1911, II, S. 1003.
[5] Lehrbuch der Nervenkrankheiten, Berlin, 1909, S. 524.
[6] Archiv. f. Psychiatrie und Nervenkrankheiten, 1908, xliv, S. 861 et seq.

follows, never precedes, delirium tremens; (4) an unambiguous instance has never come under our observation of edema occurring independently of an immediately preceding delirium, though Dana mentions the possibility of such an occurrence.

The close relationship to delirium tremens is evident, but whether wet brain is a sequela of *mania a potu*, an equivalent of the same, or an independent phase of chronic alcoholism has not been an easy problem to solve. We are strongly of the opinion, however, that delirium tremens in its classic form and wet brain represent merely successive time events in the alcoholic cycle, and that the pathological condition, wet brain, might aptly be called, clinically, comatose delirium tremens.

A long period—seven to ten years—of overindulgence in alcohol precedes the onset of the condition. This is the incubation period of delirium tremens. For some reason, as yet quite unknown, the individual develops delirium. Hypotheses are not wanting, however, to explain the transition. Among other causes suggested have been auto-intoxication, that first-aid explanation in so many doubtful conditions. According to the views of E. Meyer,[7] Ganser,[8] Ziehen,[9] and others the long alcoholic abuse renders the gastro-intestinal tract and liver incapable of destroying toxins or renders them susceptible to pathogenic organisms, with the result that the poisons accumulate and at a given time overwhelm the individual. The same view is held by some to explain the origin of liver cirrhosis in alcoholics, a theory supported by experimental work.

The hypothesis of Wagner V. Jauregg[10] is an ingenious one. It, too, is based on a toxic theory, namely, alcohol gives rise to a toxin and an antitoxin which neutralize each other as long as alcohol is exhibited. When the drug is withdrawn the antitoxin is no longer formed, with a consequent preponderance of the poison. This view is surely untenable, however, as many of our patients developed delirium after a prolonged spree, not after withdrawal of the alcohol. Starvation with its sequelæ, as seen in other conditions, is also advanced as an explanation, and has much in its favor, for it is well known that the alcoholic can take practically no food while he is drinking and, further, that the taking of nourishment goes hand in hand with improvement.

We shall not tarry longer on this interesting topic, as it does not concern itself immediately with the wet-brain condition. The predisposing factors of delirium tremens—pneumonia, trauma, especially fractures of the long bones, mental shock, combined with with a period of excessive alcoholism or a sudden withdrawal of the drug, these are all too well known to be dwelt upon.

[7] Ursachen der Geisteskrankheiten, Jena, 1907, S. 138.

[8] Zur Behandlung des Delirium Tremens, Münch. med. Woch., 1907, No. 3., S. 122.

[9] Psychiatrie, Leipzig, 1902, 2 Aufl.

[10] Quoted by Wassermeyer, q.v.

Delirium tremens shows itself in several forms. In the undeveloped type, or *forme fruste*, the so-called "touch of the horrors," the patient exhibits a vague uneasiness, starts at slight noises, may have ill-formed hallucinations of sight and hearing, is restless, loses sleep, has disturbed dreams, paresthesias, marked tremor of the hands and tongue, and a constant fear that some evil will overtake him.

The incipient period has a variable duration, usually two to three days, yielding to appropriate therapy—a good hypnotic, followed by sleep—or going over into the fully developed delirium tremens. The latter with its well-known features we shall not dwell upon except to bring to your attention a further subdivision into types which we observe with unequal frequency. These types are more or less comparable in their manifestations to the two ways in which excessive heat affects men, namely, heat exhaustion and thermic fever. In the first form, occurring in about one-fourth of all, and showing the highest mortality, there is profound asthenia, pallor, cold skin, a rolling of the eyes from side to side, and often subnormal temperature; whereas in the more frequent second type there is observed a flushed face, a full-bounding pulse, great mental and motor excitement, and possibly fever. The former requires heat and analeptica, the second cold packs and sedatives; both demand strychnin to replace the alcohol. The deciding factor as to which of these sub-types of delirium will occur is possibly the previous integrity of the cardiovascular system.

Delirium tremens, when treated, ordinarily lasts three to eight days, perhaps oftenest five, though some cases tend to continue, with clear intervals, for several weeks. In about 10 to 15 per cent., however, there is an entirely different outcome and the condition called wet brain or, better, comatose delirium tremens supervenes.

The transition from delirium to edema is fairly well marked. Striking is the semicoma succeeding the active delirium. The latter is now of a low muttering type, the individual lies with his eyes closed and is aroused with difficulty. Supraorbital pressure and rubbing of the knuckles briskly over the chest in the mid-axillary line bring about a momentary lifting of the stupor, but questions will not elicit intelligent answers. During the early stages he will swallow food and still retain consciousness enough to have delusions and hallucinations resembling a low typhoid delirium. The pulse is rapid and weak; the temperature elevated a degree or more. Conjunctivitis is common, the tongue and teeth are sordes-covered, the breath is foul, and albumin is usually present in the urine.

The train of symptoms peculiarly indicative of wet brain are essentially those of meningeal irritation and of cerebral compression. Hyperesthesia is one of the most marked and constant features; the integument is ultrasensitive, and pressure on the muscles causes

the patient to grimace and moan and try to draw himself away. Photophobia, however, is absent; as the individual does not complain we can form no judgment as to the presence of headache. Vomiting is not observed.

Rigidity in its various manifestations is always prominent, occupying with hyperesthesia the centre of the wet-brain picture. The degree to which it is present is significant of the severity of the particular case; thus Dana remarks that when the neck becomes stiff the outlook is hopeless. Kernig's sign is always present to some extent; toward the end it becomes marked.

The deep reflexes are early increased, as is the rule in delirium tremens; later, with the increase in the edema, they are lost. The pupils are usually small, equal, regular, responding sluggishly to light; they dilate somewhat if the patient can be aroused, narrowing down once more as he relapses into coma. In a small percentage of cases we have observed one or all of the criteria of the Argyll-Robertson phenomenon—usually the reflex rigidity—a condition which other observers, notably Nonne,[11] Uhthoff[12] have described in long-standing alcoholism. It is a mooted question whether this pupillary condition is permanent or not.

We are not yet prepared to express ourselves definitively as to the cerebrospinal fluid in wet brain. Cytoanalysis gives normal results; globulin and Wassermann reactions, in frank cases, are negative. It is in regard to the pressure under which the fluid exists that we withhold our opinion. Dana in his cases seems to have found a high tension the rule. He does not state, however, the means he employed to determine the pressure. As will be brought out later, there was no indication at autopsy in our cases that the cerebrospinal fluid *intravitam* was under increased tension. Clinically we have made no manometric observations, judging the pressure only by the velocity of flow accurate enough, perhaps, in a relative way. The fluid does not spurt out, as in meningitis, for example, but comes out drop by drop, with a rapidity not much different from normal. The many pathological changes recorded as characteristic of the fluid in the hybrid serous meningitis are regularly absent. In brief if the fluid is not free from abnormalities an uncomplicated case of wet brain is not present.

Side by side with the meningeal picture, except in very rare instances, to which we shall refer below, is another, not an integral part of the wet brain, but pointing rather to one of the infectious complications so regularly accompanying alcoholism. The commonest of these by far is pneumonia, usually patchy in character,

[11] Klinische und anatomische Untersuchung eines Falles von isolierter echter reflectorischer Pupillenstarre Ohne Syphilis, bei alcohol chron. gravis-Neurolog. Zentralblatt, 1912, xxxi, 60.

[12] Graefe-Saemisch-Handbuch der Augenheilkunde, Leipzig, 1901, 2 auf. Band xi, abt. 2a. S. 24.

and accounting for the temperature, tachycardia, tachypnea, leukocytosis and the like. This associated infection clouds the wet-brain picture and demands extreme caution in making a diagnosis. Neither Dana nor Lambert has dwelt at any length upon this interweaving of conditions, and it has taken us several years to feel assured that there is a meningeal phase of delirium tremens, and that it is not a manifestation of the co-existing infection.

In six instances no gross pathological lesion was found to account for the symptoms of some infection in the wet-brain complex. It has occurred to us that these may be examples of what Magnan[13] has called delirium tremens febrile. The possibility, however, of a microscopic bronchopneumonia or of some unrecognized septic condition must be admitted. Temperature and other signs of infection are hardly compatible with an uncomplicated wet brain.

All of our deaths are made "coroners' cases." Since July, 1911, in practically all instances, Dr. E. R. Le Count has performed the postmortem examinations, and to him we are indebted for the following:[14]

"As a rule, edema of the brain in delirium tremens is characterized by two features, which, although they do not serve to distinguish it invariably as to etiology, are, nevertheless frequently helpful in determining the cause of death; for without knowledge of the clinical conditon, it may be difficult to conclude from any postmortem examination that death was due to delirium tremens.

"The pia-arachnoid membranes are lifted away from the brain over the vertex, both in front and behind, but principally anterior to a line passing over the head between the external auditory meati. The fluid, as it is seen under these transparent membranes, appears slightly yellowish. In a glass pipette it is quite c[illegible] and colorless; naturally the fluid is most abundant opposite the sulci, but in many instances it is so plentiful that the arachnoid, or the outermost portion of the pia-arachnoid is lifted away from the surface or highest part of the convolutions.

"The fluid is, strictly speaking, in the loose meshwork which connects the arachnoid with the pia, for when the outer layer is broken or torn away and the fluid released there still remains the vessel-bearing part of the pia covering the brain.

"The other feature is more noticeable after the brain is removed. It is a widening of the sulci and a narrowing of the convolutions, principally of the frontal and parietal lobes. This may be general for these regions, but often there are places where pits with dimensions of 2 to 3 cm. and 0.5 cm. in depth occur—pits into which

[13] Alzheimer, Das Delirium alcoholicum febrile Magnan. Centralblatt f. Nervenheilkunde und Psychiatrie, 1904, S. 437.

[14] Personal communication.

about one-half of the terminal phalanx of the little finger may be laid. These pits, or more marked and localized regions of atrophy, are at the meeting-points of the sulci.

"There is a more or less prevalent idea in medical literature and circles that a fibrous lepto-meningitis in patches, may result from the long-continued use of alcoholic beverages. I have not found it with any regularity in the brains of persons dying of delirium tremens, and it certainly is not as characteristic as the two features emphasized.

"Another change is a softening or increased moisture of the brain tissue; but this, too, is inconstant and in some measure probably connected with cadaveric decomposition. In some instances the finer arterioles of the pia are more engorged with blood than in others; this apparently is associated with a type of active or maniacal delirium rather than with those showing a lethargic or semicomatose delirium. The atrophy of the cerebral cortex and the accumulation of fluid have impressed me as an edema *ex vacuo;* and, certainly, there is no indication at the postmortem examination that the fluid during life is under an increased pressure."

Wassermeyer nowhere makes mention of a cerebral edema similar to the above. Dana, on the other hand, who has autopsied twenty of his own cases of wet brain, found such a condition, and from it coined the name. It is most marked, according to him, in the arachnoid and subarachnoid spaces, and may penetrate into the brain substance for a variable distance. An increase in the ventricular fluid content is also not infrequent, with consequent dilatation of the ventricular spaces. As for the remainder, Dana's findings are not different from those of delirium tremens. The picture of the latter, we digress to say, is by no means a constant one, as Wassermeyer points out. He emphasizes the hyperemia, the frequent capillary, less often extensive, hemorrhages; the occasional thickening of the meninges; and in some instances the presence of a condition indistinguishable from the poliomyelitis acuta hemorrhagica superior of Wernicke and in others from a pachymeningitis hemorrhagica interna. Finally there may or may not be evidence of degeneration in the nerve cells or a growth of the glial tissue.

It would seem, therefore, that the more or less extensive fluid accumulation in the arachnoid, with the widening of the sulci and the narrowing of the convolutions, constitute the salient autopsy findings of wet brain. As there is no evidence of the fluid's having existed under increased pressure, it might be urged that the primary change is cerebral atrophy and that the fluid is secondary, much like the *vacatwucherung* about the kidneys when the latter atrophy and the space they formerly occupied is filled with a compensatory fatty deposit.

The diagnosis of alcoholic cerebral edema may be extremely

easy or, on the other hand, impossible. When there has been opportunity to observe the patient through the phases of incipient and fully developed delirium tremens there is, as a rule, no difficulty; the same is to a great extent true in cases which are brought into the hospital already suffering from frank delirium tremens, especially when there is no history of trauma. At times the comatose form of delirium tremens may have to be distinguished from the ordinary form (especially from the sub-type we have called asthenic); the semicoma in the former, the hyperesthesia and rigidity, the absence of the rolling of the eyes, etc., usually serve to differentiate the two; in addition following wet brain there is usually a total amnesia, while after delirium tremens the individual commonly recognizes part of his hallucinations, as such, or he may jumble hallucinations and facts.

A history of, or the possibility of, trauma may cloud the diagnosis almost beyond the possibility of solution. With us basal skull fracture is the condition above all others to be kept in mind, as bitter experience has shown time and again. The following points have been of the greatest value in the diagnosis of basal fractures, in the absence of cerebrospinal fluid or blood from the ear or nose; delayed ecchymoses over the mastoid and about the orbit; eye-muscle palsies; paretic involvement of one or more extremities; and blood in the spinal fluid.

A number of other conditions must likewise be taken into account. The meningitides can, usually, be ruled out by the negative bacteriological, serological, and chemical cerebrospinal fluid tests, according to the type present. Uremic coma, which may present great difficulties both because of a marked similarity to wet brain on the clinical side and also because most alcoholics show renal changes, is recognized by the associated cardiovascular phenomena, retinal changes, by the exaggerated knee-jerks usually present, the absence of hyperesthesia, and the occurrence of convulsions and vomiting. Cerebrospinal lues is distinguished by the Wassermann and globulin reactions, by cyto-analysis in the spinal fluid, and by the presence of definite organic changes in the nervous system Cerebral hemorrhage, thrombosis, and embolism are suggested by etiology and by the organic nervous findings. Finally, pachymeningitis, hemorrhagica interna, and polioencephalitis acuta superior of Wernicke must be thought of because of the common etiology—alcohol. In these, however, gross nervous changes are distinguishing characteristics.

The duration of wet brain is variable, the condition lasting from two to twelve weeks, usually about three. The mortality is nearly 75 per cent.; death may occur at any time, oftenest from a bronchopneumonia, which frequently can scarcely be diagnosed in view of the paucity of physical signs. Unfavorable symptoms, indicative of an increase in the edema, are augmented muscular rigidity

especially of the neckmuscles, diminution in the size of the pupils and more marked sluggishness in their response to light, and deepening of the coma. The pulse finally becomes rapid and feeble, there is incontinence of urine and feces, and death occurs.

As to treatment, aside from symptomatic measures, little can be done. The death rate would indicate this. Cardiac stimulants are in order. The nutrition of the patient suffers markedly consequent upon the long state of coma; persistent effort should be made to force liquids; and in the severer cases it may be necessary to give alimentation by the nasal tube. Elimination must also not be neglected. Ergot we have found harmful as compared with its usefulness in the asthenic type of delirium tremens. Lumbar puncture has been of no service in our hands, contrary to favorable reports by Dana. Scientific hydrotherapy, now not available to us may prove an important addition to our therapeutics.

RESUMÉ. After long-continued overindulgence in alcohol an individual, for reasons as yet undetermined, develops delirium tremens. The latter usually manifests itself in three stages: incipient, fully developed (classic), and comatose (wet brain). The symptoms of wet brain are essentially meningeal, semicoma, generalized hyperesthesia, and muscular rigidity (Kernig and neck rigidity) standing out prominently; the more marked are the latter two features, the graver the prognosis. The cerebrospinal fluid is, to all appearances, normal. The mortality is nearly 75 per cent. Necropsy reveals no gross lesions aside from the more or less marked fluid accumulation in the pia-arachnoid space, a widening of the sulci, and a narrowing of the convolutions to account for the symptoms of changes in the brain. Associated with the cerebral edema complex there is very often bronchopneumonia, which clouds the diagnosis and usually is responsible for death. The differential diagnosis must concern itself particularly with the possibility of a concomitant skull fracture which may easily be obscured by the nervous manifestations of comatose delirium tremens.

THE STUDY OF TWO HUNDRED AND FIFTY STAINED BLOOD-FILMS IN PYORRHEA ALVEOLARIS.

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THE object of this paper is not to discuss the etiology or the prognostic value of the stained blood-film. It is written for the purpose of informing the reader that tinctorial changes and an increase of the large lymphocytes are common findings in the stained blood-films made from patients affected with pyorrhea alveolaris.